Childhood Socioeconomic Status in Predicting Metabolic Syndrome and Glucose Abnormalities in Adulthood: The Cardiovascular Risk in Young Finns Study

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OBJECTIVE

We prospectively examined whether family socioeconomic status (SES) in childhood is associated with metabolic syndrome (MetS), impaired fasting glucose (IFG), or type 2 diabetes in adulthood.

RESEARCH DESIGN AND METHODS

The sample comprised 2,250 participants from the longitudinal Cardiovascular Risk in Young Finns Study cohort. Participants were 3–18 years old at baseline (mean age 10.6 years), and they were followed for 31 years. SES was characterized as reported annual income of the family and classified on an 8-point scale.

RESULTS

For each 1-unit increase in family SES in childhood, the risk for adult MetS decreased (risk ratio [95% confidence interval] 0.94 [0.90–0.98]; *P* = 0.003) when adjusted for age, sex, childhood cardiometabolic risk factors (lipids, systolic blood pressure, insulin, and BMI), childhood physical activity, and fruit and vegetable consumption. The association remained after adjustment for participants' own SES in adulthood (0.95 [0.91–0.99]; *P* = 0.005). A similar association was seen between childhood SES and the risk of having either adult IFG or type 2 diabetes (0.96 [0.92–0.99]; *P* = 0.01, age and sex adjusted). This association became nonsignificant after adjustment for childhood risk factors (*P* = 0.08). Of the individual components of MetS, lower SES in childhood predicted large waist circumference (0.96 [0.93–0.99]; *P* = 0.003) and a high triglycerides concentration (0.96 [0.92–1.00]; *P* = 0.04) after adjustment for the aforementioned risk factors.

CONCLUSIONS

Lower SES in childhood may be associated with an increased risk for MetS, IFG, and type 2 diabetes in adulthood. Special attention could be paid to children of low SES families to decrease the prevalence of MetS in adulthood.

Metabolic syndrome (MetS) is a cluster of cardiometabolic risk factors that predisposes individuals to a fivefold increased risk for type 2 diabetes and a twofold increased risk for cardiovascular disease (1). Several studies have shown that MetS predicts cardiovascular disease after adjustment for its individual components, indicating the importance of the clustering of risk factors (2,3). In developed

countries, MetS is present in ~25% of the adult population, and because of the global increase in obesity and sedentary lifestyle, the prevalence of MetS continues to increase (4).

The Whitehall study, begun in 1967 among British male civil servants, was the first to document an inverse association between socioeconomic status (SES) and mortality (5). In the Whitehall II study, a similar gradient in mortality was shown among female civil servants (5). A study addressing childhood determinants of ideal cardiovascular health showed that the prevalence of MetS components is increased in lower socioeconomic groups (6). Several systematic reviews have reported an inverse association of SES with obesity and type 2 diabetes linked with MetS (7,8), but no recent prospective study has shown an association between childhood SES and MetS later in adulthood for the general population. Prospective data on the association between childhood SES and type 2 diabetes also are lacking.

Using data from the longitudinal Cardiovascular Risk in Young Finns Study cohort, we examined the independent association of childhood family SES at age 3–18 years on MetS and type 2 diabetes 27–31 years later in adulthood (n = 2,250). The results provide novel data that identify families whose children have a greater risk for developing MetS or type 2 diabetes later in life.

RESEARCH DESIGN AND METHODS

Participants

The Young Finns Study is an ongoing population-based follow-up study of atherosclerosis precursors in Finnish children and young adults. The first cross-sectional survey was conducted in 1980 in 3,596 participants aged 3–18 years (12). These participants were randomly chosen from the national register of the five study areas. Thereafter, several follow-up surveys have been performed (9). The two latest follow-up surveys were completed in 2007 and 2011 by 2,204 and 2,036 participants from the original cohort, respectively. In this study, the sample comprised 2,250 participants aged 3–18 years at baseline who provided data on SES and cardiometabolic risk factors in childhood (1980) and adulthood. In adulthood, data from the 2011 survey (n = 1,927) were primarily used in the analyses. If complete data were not available from 2011, then data from the 2007 survey were used (n = 323 [14.3%]). Thus, the mean follow-up period was 30.4 years. In additional analyses that examined the life-course levels of MetS components, data from the 1986 and 2001 surveys on participant SES and cardiometabolic risk factors were used. Participants with type 1 diabetes or who were pregnant were excluded from the analyses. All participants provided written informed consent, and the study was approved by local ethics committees (University of Turku and University of Tampere).

Clinical Characteristics and Laboratory Measures

Height, weight, and waist circumference were measured at all examinations (9). BMI was calculated as weight in kilograms divided by height in meters squared. Blood pressure was measured from the right-side brachial artery with a standard mercury sphygmomanometer in 1980 and with a random-zero sphygmomanometer (Hawksley & Sons Ltd.; Lancing, U.K.) in 1986, 2001, 2007, and 2011 (10). Blood pressure was measured in the sitting position after a 5-min rest. The average of three measurements was used in the analysis.

Venous blood samples were collected after a 12-h fast. Lipid determinations for triglycerides, total cholesterol, and HDL cholesterol were done with standard methods (11). LDL cholesterol was calculated with the Friedewald equation for participants with triglycerides <4 mmol/L. Plasma glucose concentrations were analyzed enzymatically, and serum insulin was measured by microparticle enzyme immunoassay kit. Details of the methods have been described previously (11).

Physical Activity and Diet

Data on dietary habits and physical activity in childhood were obtained through questionnaires (11). A physical activity index comprising the duration, intensity, and frequency of physical activity, including leisure-time physical activity and commuting, was calculated to indicate habitual physical activity (12,13). Separate questionnaires were used for the younger (3–6 years of age, a parent-completed questionnaire) and older (9–18 years of age, a self-completed questionnaire with the help of parents, if needed) children. To ensure that the data could be compared between the two age-groups, the calculated physical activity indices were age standardized. To examine the frequency of vegetable and fruit consumption, participants (or the parents of children 3–6 years of age) completed a questionnaire on habitual dietary choices during the past month through six response categories: 1 = daily, 2 = almost every day, 3 = a couple of times per week, 4 = about once a week, 5 = a couple of times per month, and 6 = more seldom. The response categories were converted into times of consumption per week. Detailed information on the food frequency questionnaires and their validity has been published elsewhere (14,15).

Classification of SES

Annual income was considered an indicator of SES in both childhood and adulthood (16). Annual family income strata at the time of enrollment were determined as follows: category 1, <15,000 Finnish marks (FIM) (corresponding to ~€7,050 in 2011); category 2, 15,001–25,000 FIM; category 3, 25,001–35,000 FIM; category 4, 35,001–45,000 FIM; category 5, 45,001–55,000 FIM; category 6, 55,001–75,000 FIM; category 7, 75,001–100,000 FIM; and category 8, >100,000 FIM. Participant's income in adulthood in 2007 and 2011 was also classified on an 8-point scale, ranging from annual gross income from 1 (<€10,000) to 8 (>€70,000) (17). In a sensitivity analysis, we also defined childhood SES according to parental years of education.

Classification of MetS, Impaired Fasting Glucose, and Type 2 Diabetes

MetS was defined by using the harmonized criteria proposed in 2009 (18). Data from the follow-up conducted in 2011 were primarily used. If the participant did not attend the 2011 follow-up, data from the 2007 follow-up were applied to indicate adult MetS. MetS was diagnosed if the participant had at least three of the following five components: *1*) waist circumference ≥102 cm for males and ≥88 cm for females, *2*) triglycerides ≥1.7 mmol/L (≥150 mg/dL) or specific treatment for this lipid abnormality, *3*) HDL cholesterol <1.0 mmol/L (<40 mg/dL) in males or <1.3 mmol/L (<50 mg/dL) in females or specific treatment for this lipid abnormality, *4*) blood pressure ≥130/85 mmHg or treatment of previously diagnosed hypertension, and *5*) fasting plasma glucose ≥5.6 mmol/L (≥100 mg/dL) or specific drug treatment

of elevated glucose. In total, 481 participants had MetS in adulthood.

Impaired fasting glucose (IFG) was defined as having a fasting plasma glucose ≥5.6 mmol/L (19). IFG was found in 564 participants. Participants were classified as having type 2 diabetes if they had a fasting plasma glucose ≥7.0 mmol/L (≥126 mg/dL) in accordance with American Diabetes Association criteria (19), had an HbA_{1c} level of ≥6.5% (≥48 mmol/mol), reported receiving oral hypoglycemic agents and/or insulin injections, and did not have type 1 diabetes or reported a diagnosis made by a physician. Type 2 diabetes was found in 81 participants. In line with MetS, data from the 2011 follow-up were primarily used and in the case of missing data, complemented data obtained in 2007. Separate analyses were done for the group of participants with type 2 diabetes and for a combined group of participants with either IFG or type 2 diabetes.

Statistical Analyses

To examine differences between characteristics of males and females, we used age-adjusted linear regression for continuous outcomes and logistic regression for binary outcomes. Continuous variables were described by mean ± SD and binary variables as percentages. To study associations of family SES in childhood with MetS, IFG, type 2 diabetes, and components of MetS (categorical variable) in adulthood, we examined risk ratios (RRs) using Poisson logistic regression. The analyses were adjusted only for age and sex (model 1); for age, sex, conventional childhood cardiometabolic risk factors (lipids, systolic blood pressure, insulin, and BMI), childhood physical activity, and fruit and vegetable consumption (model 2); and further for SES in adulthood (model 3). To study the association of intergenerational mobility of SES on the prevalence of MetS in adulthood, the study population was classified into four groups according to SES status in childhood and adulthood as follows: group 1 (n = 501), SES under the median in both childhood and adulthood; group 2 (n = 541), SES above the median in childhood and below the median in adulthood; group 3 (n = 421), SES below the median in childhood and above the median in adulthood; and group 4 (n = 787), SES above the median in both childhood and adulthood. The differences in the prevalence of MetS in these SES groups were examined using cross-tabulation together with age- and sex-adjusted logistic regression. We also compared risk factor profiles in childhood and adulthood among these SES groups by using multiple comparisons separately for every risk variable. Finally, we analyzed the life-course levels of components of MetS in these groups, using multiple comparisons adjusted for age and sex to the mean values in 4–5 time points in the groups. We used the Tukey-Kramer method to compute multiplicity-adjusted P values. All statistical tests were performed using SAS 9.4 software (SAS Institute, Cary, NC), with statistical significance inferred at a two-tailed $P < 0.05$.

RESULTS

The baseline characteristics of the study population are shown in Supplementary Table 1. At baseline, the participants were, on average, 10.6 years old, and slightly more females than males comprised the study cohort. Females had a higher LDL cholesterol concentration, whereas systolic blood pressure was higher in males. When sex by MetS-type 2 diabetes interactions were studied, no significant sex differences were detected, indicating that the effect of SES on MetS and type 2 diabetes was similar between males and females. Thus, the sexes were analyzed together.

Higher SES in childhood was associated with a lower risk for MetS in adulthood (RR 0.93 [95% CI 0.90–0.97]; P = 0.0003, adjusted for age and sex) (Fig. 1). In the additional analysis using parental years of education as an indicator of childhood SES, the results were similar (RR 0.97 [95% CI 0.95–0.99]; P = 0.02, adjusted for age and sex). The association remained significant after adjustment for conventional cardiovascular risk factors, including physical activity and fruit and vegetable consumption in childhood (RR 0.94 [95% CI 0.90–0.98]; P = 0.003). These results persisted when the analysis was also adjusted for the participants' own SES in adulthood (RR 0.95 [95% CI 0.91–0.99]; P = 0.005).

Childhood SES was also inversely associated with the components of MetS (Table 1, model 1, adjusted for age and sex). Except for blood pressure, these associations persisted after adjustment for cardiovascular risk factors in childhood (model 2). When also adjusted for their own SES in adulthood (model 3), the only statistically significant associations between childhood SES and the MetS components were found for waist circumference and triglyceride levels.

Prevalence of MetS in adulthood was compared among the four childhood-adulthood SES groups (Fig. 2). The prevalence of MetS was lowest in group 4 (high SES in both childhood and adulthood) compared with the other groups, including group 3 (low SES in childhood but high in adulthood).

To gain more insight into the highest prevalence of MetS in group 3, we examined individual risk factors in childhood and adulthood by comparing group 3 with the other groups (Table 2). The analyses revealed that group 4 participants had a more-favorable risk factor profile. In addition, we analyzed the life-course levels of MetS components in the childhood-adulthood SES groups (Supplementary Figs. 1–6). Differences were seen among the groups throughout the life course in every MetS component, particularly highlighting more favorable levels in group 4.

In addition to MetS, we studied the association of childhood SES with later risk for IFG and type 2 diabetes. In line with MetS, childhood SES was inversely associated with the combine variable (i.e., having either IFG or type 2 diabetes) (RR 0.96 [95% CI 0.92–0.99]; P = 0.01, adjusted for age and sex). The association was attenuated after adjustment for conventional cardiometabolic risk factors in childhood (RR 0.97 [95% CI 0.93–1.00]; P = 0.08). When type 2 diabetes was analyzed separately, no association was found between childhood SES and type 2 diabetes in adulthood (RR 0.92 [95% CI 0.82–1.02; P = 0.10, adjusted for age and sex] and 0.95 [95% CI 0.85–1.06; P = 0.34, adjusted for age, sex, and conventional cardiometabolic risk factors in childhood], respectively).

CONCLUSIONS

This prospective study indicates that higher SES in childhood is associated with a lower risk for MetS >30 years later in adulthood. The relation was independent of childhood conventional cardiometabolic risk factors and participants' own SES in adulthood. Similar results were seen between childhood SES and risk of having IFG or type 2 diabetes later in life, although the association

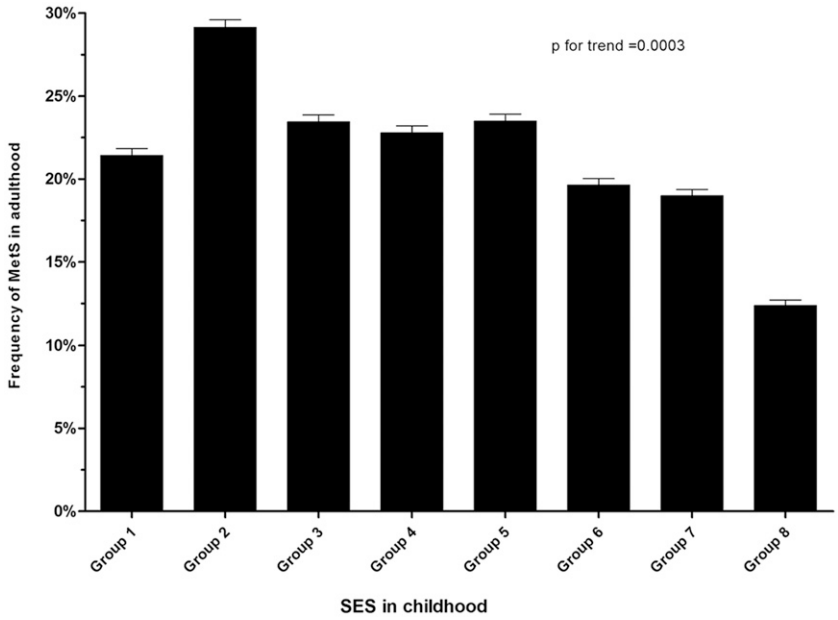

Figure 1—Prevalence of MetS in various childhood SES groups (n = 2,250). Data are mean ± SD.

was attenuated after adjustment for cardiometabolic risk factors in childhood. The lowest prevalence of MetS (18.7%) was found in those who maintained their SES above the median from childhood to adulthood. Those who had an SES below the median in childhood but above it in adulthood had the highest prevalence (25.4%) of MetS in adulthood.

The first documented association between SES and mortality was reported in the Whitehall studies that begun in 1967 (5). After that, the inverse relation between SES and risk factor levels of various chronic diseases has been found across populations (8,20). Previous studies also have shown an association between SES and the risk of MetS in adulthood (21,22). A study among 9,746 Chinese adults aged >50 years showed that high SES is inversely associated with the prevalence of MetS in women but not in men (21). A similar result was found in the Atherosclerosis Risk in Communities study among >10,000 men and women aged 45–64 years (22). In this study, no SES- sex interaction was found, indicating that the association between childhood SES and MetS is similar in males and females. Few studies have examined whether mostly retrospectively assessed SES in childhood is associated with the risk of MetS in adulthood and have revealed inconclusive results (23–26). In most of these studies, no associations were found between childhood SES and adult MetS, or they did not persist after taking into account adult SES. An inverse association between retrospectively assessed childhood SES and MetS at baseline when participants were 42–52 years old was reported in the Study of Women's Health Across the Nation (23). In addition, participants' own education was associated with MetS after 12 years of follow-up, whereas childhood SES was not significant in predicting MetS. Thus, the impacts of childhood SES on the risk of having MetS in adulthood remain poorly understood in data representative of the general population and when several childhood cardiometabolic risk factors and one's own adult SES are taken into account. Our previous studies have shown that SES in childhood is inversely associated with cardiovascular risk factors and vascular structure and function (6,27). The current prospective study thus provides novel data on the independent association between SES in childhood and the risk of having MetS three decades later in adulthood.

To the best of our knowledge, the effect of intergenerational mobility in SES on MetS in adulthood has not been investigated previously. In a study where childhood SES was determined by parental educational status, parent SES had no association with having a healthy lifestyle as an adult when adjusted for participants' own SES; only the SES they finally attained was significant (28). In the current study, intergenerational mobility had an impact on the prevalence of MetS in adulthood, as indicated, for example, by both upwardly and downwardly mobile SES groups differing from the group that stayed above the SES median in childhood and adulthood. Of note, participants who experienced an increase in SES from childhood did not show a reduced risk for MetS but rather, had the highest prevalence, suggesting that an increase in SES does not confer protective effects against MetS but instead, predisposes to a higher risk for MetS in adulthood. After adjustment for sex and age, the prevalence was statistically higher compared with having an SES that is high in both childhood and adulthood. In addition, participants who obtained a higher SES in adulthood had more adverse levels in

Table 1—Relative risk for components of MetS in adulthood according to family SES in childhood (n = 2,250)

Adulthood variable	Percentage	Model 1	P value	Model 2	P value	Model 3	P value
High waist circumference	34.8	0.96 (0.93–0.98)	0.002	0.95 (0.93–0.98)	0.001	0.96 (0.93–0.99)	0.003
High blood pressure	21.7	0.96 (0.92–1.00)	0.04	0.97 (0.94–1.01)	0.19	0.97 (0.94–1.01)	0.20
Low HDL cholesterol	34.0	0.95 (0.93–0.98)	0.0007	0.97 (0.93–1.00)	0.03	0.97 (0.94–1.00)	0.06
High triglycerides	20.6	0.94 (0.90–0.98)	0.002	0.95 (0.92–1.00)	0.03	0.96 (0.92–1.00)	0.04
High fasting plasma glucose	25.1	0.96 (0.92–0.99)	0.01	0.96 (0.93–1.00)	0.05	0.97 (0.94–1.01)	0.15

Data are RR (95% CI) unless otherwise indicated. RRs and P values were calculated using Poisson logistic regression. Relative risks are for a 1-unit increase in family SES in childhood. Adjusted model 1 included age (3–18 years at baseline) and sex. Adjusted model 2 included model 1 covariates plus childhood LDL cholesterol, HDL cholesterol, triglycerides, systolic blood pressure, BMI, insulin, frequency of fruit and vegetable consumption, and physical activity index. Adjusted model 3 included model 2 covariates plus participant's own SES (annual income) in adulthood.

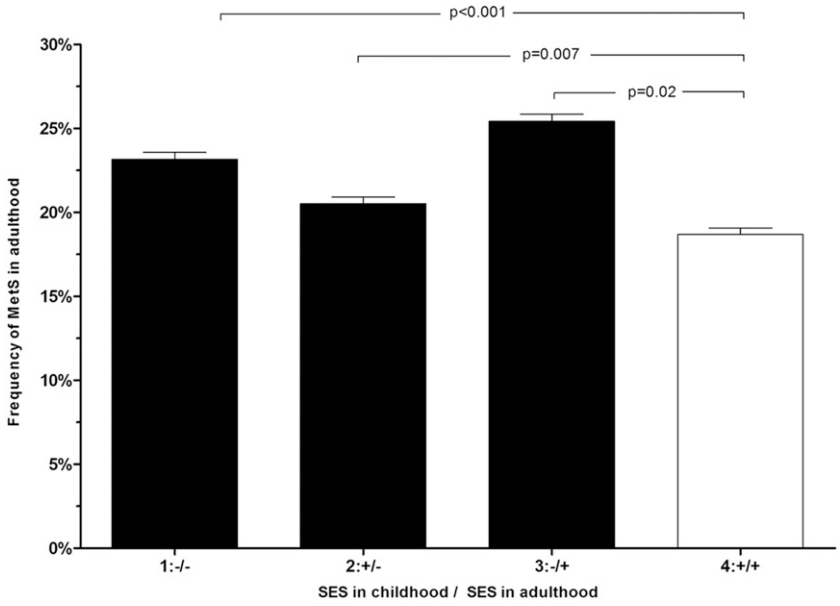

Figure 2—Prevalence of MetS in various childhood and adulthood SES groups (n = 2,250). Data are mean ± SD. −, below the median SES; +, above the median SES.

nearly all MetS components in adulthood than those who remained in the high SES group from childhood to adulthood. This finding indicates that low childhood SES has a residual effect on MetS components in adulthood above the effect of the participant's own SES. With these data, we could not provide definite mechanisms behind the increased risk of MetS with an increase in SES from childhood to adulthood; thus, further research on this effect is warranted. Moreover, when the differences between the groups in every MetS component were compared throughout the life course, risk factors among those with constantly high SES were more favorable, further suggesting the benefits of sustained high SES.

Multiple mechanisms underlie the associations between childhood SES and adult MetS and may have complex interrelations. The pathophysiological basis of MetS is insulin resistance, which is strongly associated with abdominal obesity (1,29). On the other hand, abdominal obesity has a strong inverse relationship with SES (8). In a South Korean study by Lim et al. (30), income differences were associated more strongly with obesity, hypertension, and dyslipidemia components of MetS compared with hyperglycemia. The finding is in line with the current data, showing significant associations between family income and subsequent MetS but not with elevated glucose levels. A recent study showed that SES is inversely associated with fruit and vegetable consumption among all Nordic countries (31), and lower SES relates to physical inactivity (32). Furthermore, the relationship of dietary habits and physical activity with MetS and its components is well established (14,33,34). In the current study, the association of childhood SES with MetS persisted after adjustments for fruit and vegetable consumption, physical activity, and several other conventional childhood cardiometabolic risk factors, suggesting that other mediating factors may be at play that affect the association.

One possible underlying mechanism is the stress-mediated pathway. Lower SES has been shown to associate with greater stress hormone levels, catecholamines, and cortisol (35). Stress modifies fat metabolism and, subsequently, insulin sensitivity and predisposes to poor diet and a sedentary lifestyle, thereby contributing to the development of MetS (36,37). Moreover, infection could be a possible agent between childhood SES and MetS because childhood infection has been associated with a worse cardiovascular risk factor profile, especially among low-SES families (16). Finally, family SES might be linked to the development of MetS through its impact on birth weight related to intrauterine nutrition; malnutrition of fetus and low

Table 2—Risk factors in childhood and adulthood in various SES groups compared with group 3

	Group 3	Group 1	Group 2	Group 4	*P* value*	*P* value†	*P* value‡
Risk factors in childhood							
BMI (kg/m^2)	18.2 (3.0)	17.8 (3.1)	17.8 (3.0)	17.8 (2.9)	1.00	0.35	0.63
Systolic blood pressure (mmHg)	114.5 (12.1)	111.8 (12.6)	111.6 (12.0)	112.2 (11.3)	0.32	0.61	0.04
HDL cholesterol (mmol/L)	1.54 (0.31)	1.54 (0.31)	1.58 (0.29)	1.58 (0.31)	0.96	0.54	0.13
Triglycerides (mmol/L)	0.69 (0.34)	0.70 (0.32)	0.66 (0.30)	0.63 (0.29)	0.98	0.42	0.006
Insulin (mU/L)	10.10 (6.36)	9.86 (5.98)	9.81 (6.00)	9.44 (5.75)	1.00	1.00	0.63
Risk factors in adulthood							
BMI (kg/m^2)	27.0 (4.9)	27.0 (5.7)	26.4 (5.2)	25.8 (4.5)	0.82	0.92	0.007
Waist circumference (cm)	93.6 (13.9)	91.5 (15.2)	90.5 (14.6)	90.6 (13.3)	0.85	1.00	0.02
Systolic blood pressure (mmHg)	122.4 (14.7)	119.8 (14.7)	117.9 (14.3)	118.2 (13.4)	1.00	0.43	<0.0001
HDL cholesterol (mmol/L)	1.27 (0.34)	1.32 (0.31)	1.36 (0.32)	1.34 (0.35)	0.91	0.86	0.02
Triglycerides (mmol/L)	1.48 (0.98)	1.37 (1.27)	1.32 (1.61)	1.28 (0.83)	1.00	1.00	0.06
Fasting plasma glucose (mmol/L)	5.37 (0.59)	5.43 (1.03)	5.34 (0.69)	5.32 (0.74)	0.006	0.24	0.94

Data are mean ± SD. *P* values are for Tukey-Kramer–adjusted multiple comparisons. Group 1 (n = 501), SES below the median in childhood and adulthood; group 2 (n = 541), SES above the median in childhood and below the median in adulthood; group 3 (n = 421), SES below the median in childhood and above the median in adulthood; group 4 (n = 787), SES above the median in childhood and adulthood. *Compared with group 1; †compared with group 2; ‡compared with group 4.

birth weight may lead to reduced insulin sensitivity, which favors the occurrence of hypertension, insulin resistance, and hypercholesterolemia later in life (38).

We acknowledge that this study has limitations. First, because of the rather young age of the study cohort in the latest follow-up, the prevalence of MetS, IFG, and especially type 2 diabetes was relatively low but in line with other Finnish cohorts (39). This may explain why we did not find an association between childhood SES and separately analyzed type 2 diabetes, although the RR was of similar magnitude to MetS. Second, some limitations in the classification of SES could be due to unemployment, poor health, and other life circumstances that affect income. Third, because the rates of SES mobility depend on how the mobility is defined and how the various groups of mobility are formed, the rates of mobility in this study cannot be fully compared with the other cohorts. Additionally, the generalizability of this study is limited to white populations because the study cohort was racially homogenous. Finally, possible bias due to loss to follow-up was present. However, the Young Finns Study group has been dynamic, and we have previously reported that baseline risk factors were similar among participants and nonparticipants (40). Thus, the current study population probably represents the original population. Major strengths of this study include the longitudinal design that began in childhood and continued through 31 years of follow-up and the participants being well phenotyped in both childhood and adulthood.

In conclusion, low SES in childhood is associated with a higher risk for MetS in adulthood independently of conventional childhood risk factors and one's own SES in adulthood. These results emphasize that special attention should be paid to children of low SES families with the aim to decrease prevalence of MetS and health inequalities later in life.

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